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TOXOPLASMOSIS IN WALLABIES (*MACROPUS RUFOGRISEUS* AND *MACROPUS EUGENII*): BLINDNESS, TREATMENT WITH ATOVAQUONE, AND ISOLATION OF *TOXOPLASMA GONDII*

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ABSTRACT: Australasian marsupials, especially wallabies, are highly susceptible to clinical toxoplasmosis. This paper describes the use of atovaquone for effective treatment of toxoplasmosis in 4 Bennett's wallabies (*Macropus rufogriseus*), along with the serology, isolation of *Toxoplasma gondii*, and genotyping of 3 *T. gondii* isolates from 5 captive wallabies. This is the first report of success in treating acute toxoplasmosis in wallabies, the first report of serology followed over a significant period of time, and the first report of isolation and genetic typing of *T. gondii* from wallabies in the United States.

Toxoplasmosis is a serious disease of Australasian marsupials, and there are numerous reports of deaths in zoos, including in the United States (Boorman et al., 1977; Jensen et al., 1985; Patton et al., 1986; Dubey et al., 1988; Miller et al., 1992; Adkesson et al., 2007). These animals have also died from toxoplasmosis in the wild (Attwood et al., 1975; Obendorf and Munday, 1983; Johnson et al., 1989; Canfield et al., 1990).

There is little information concerning treatment of toxoplasmosis in wallabies, and no report of successful treatment, and there is no record of recovery from an advanced stage of toxoplasmosis in wallabies.

MATERIALS AND METHODS

Background and treatment of wallabies

Ten adult wallabies (5 Tammar [*Macropus eugenii*] and 5 Bennett's [*Macropus rufogriseus*]) were imported from New Zealand. Wallabies arrived in Philadelphia, Pennsylvania, on 16 May 1996 by air within 36–48 hr of shipment. Five of the imported wallabies (2 Tammar and 3 Bennett's) died suddenly, within 2 mo of their arrival. The necropsy and histological examinations were consistent with toxoplasmosis. Two Tammar wallabies were unaffected. The remaining 3 (nos. 1–3) female wallabies (1 *M. eugenii* and 2 *M. rufogriseus*) from this shipment, and 2 male *M. rufogriseus* (nos. 4 and 5) born and raised in the United States are described in this report. The wallabies were housed in the home of C.C., who cared for them.

Wallaby no. 1, a female Tammar, was about 18 mo old and had been caught in the wild on the North Island, New Zealand. This wallaby was in poor health on arrival and was treated for intestinal parasites using ivermectin, amprolium, sulfadimethoxine, and pyrimethamine intermittently for 3 mo. Toxoplasmosis was suspected, but the eyes were not examined. The wallaby died on 14 August 1996. A necropsy examination was performed, from which tissue samples were received at the Animal Parasitic Diseases Laboratory (APDL), Beltsville, Maryland, 2 days later for isolation of *Toxoplasma gondii* (Table I).

Wallabies nos. 2 and 3 were adult female *M. rufogriseus*; had been caught in the wild on the South Island, New Zealand; and they were in good body condition on arrival in Philadelphia. Three months later, they became lethargic and were suspected to have toxoplasmosis. Wallabies nos. 2 and 3 were initially treated with pyrimethamine (Daraprim; Glaxo Wellcome Inc., Research Triangle Park, North Carolina), 1 mg/kg, oral, once daily and sulfadimethoxine (Albon; SmithKline Beecham Animal Health, West Chester, Pennsylvania), 30 mg/kg, oral, twice daily. Leucovorin (Wellcovorin, Burroughs Wellcome Co., Research Triangle Park, North Carolina), 1 mg/kg, oral, once daily was added to prevent problems from folic acid antagonism (Spielman, 1992). Vitamin E 200 international units (I.U.), oral, once daily was given to counteract stress caused by capturing and treating the wallabies (Spielman, 1992). Probiotics were given to support maintenance of normal intestinal flora.

During the first week of this treatment, the wallabies remained lethargic, progressively lost weight, developed incoordination while hopping, and began listing to the right, when standing. They had signs of progressive loss of vision with decreasing ability to perceive an approaching person, avoid collisions with stationary objects in their environment, and negotiate a flight of stairs.

Wallabies nos. 2 and 3 seemed moribund; therefore, administration of pyrimethamine, sulfadimethoxine, and leucovorin was stopped; treatment was changed to atovaquone (Mepron suspension; Glaxo Wellcome Inc.), 100 mg/kg, oral, every 24 hr, along with peanut oil, volume equal to the volume of atovaquone administered, to facilitate absorption (M. Rogers, pers. comm.). Vitamin E 200 I.U., oral, once daily, and probiotics were continued.

On the evening that atovaquone was begun, the more severely ill wallaby no. 2 had started to show signs of congestive heart failure, with bilateral wheezing and profound listlessness. These signs were worse the following morning. Auscultation of the thorax revealed bilateral rales over the lung fields, absence of breath sounds at the bases, tachypnea, and a rapid invariable heart rate. By that evening, heart and lung parameters were within normal limits, i.e., within 24 hr of starting the atovaquone.

Ophthalmic examination of wallabies nos. 2 and 3 revealed raised spots of yellow exudate, retinal damage bilaterally with splotchy pigment changes consisting of areas of atrophy interspersed with vertical light changes, and papilledema with blurring of the optic disc margins 1 day before initiation of treatment with atovaquone. Behavior was consistent with complete blindness. Papilledema resolved by day 5 of treatment with atovaquone. Eyesight was steadily improving after that time, as evaluated by the ability to evade capture, navigate a long flight of stairs, and avoid running into obstacles. Ophthalmic examination 8 wk after beginning atovaquone revealed that the retinal changes were stable. There was also evidence of early cataract formation in both eyes and slight haze in the vitreous. The optic nerves showed pallor with narrow vessels in both wallabies.

Both wallabies had developed incoordination while hopping, and they began seriously listing to starboard, when standing, before becoming blind. With atovaquone, their coordination improved quickly, but they retained a slight tilt to the right when standing at rest. This diminished from about 30°, to 5 to 10°, over 2 mo. Improvement remained at that level. After regaining eyesight, they failed to eat enough to maintain their weight. When hand fed, they ate good quantities and gave no signs of avoiding eating because of discomfort. Supplemental feeding was needed for 3 mo. Observation of their overall behavior and responsiveness to the environment was consistent with the explanation that failure to eat sufficiently was a result of cognitive impairment. The wallabies continued to have regular estrus cycles and to be bred approximately every month. Diagnosis was confirmed and followed by serology (Table II). Initially, the plan was to give atovaquone at 750 mg, oral, every 24 hr, along with 10 ml of peanut oil, for 6 mo. When the wallabies had young (joeys) in the pouch, the plan was modified. To protect the young from possible risk if infection could be acquired from their dams, atovaquone was to be continued until they were 8 mo in the pouch and could readily be removed to be hand raised. Wallaby no. 3 incidentally

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TABLE I. Isolation of *Toxoplasma gondii* from wallabies.

Wallaby no.	Species	Date of necropsy	Age at necropsy (yr)	MAT titer	Histological lesions in wallaby	No. of mice *T. gondii* positive/no. inoculated		*T. gondii* isolate designation
						Brain	Other tissues	
1	*Macropus eugenii*	16 August 1996	2	200	No data		10/10*	Tg WyUs 1
2	*Macropus rufogriseus*	1 March 1999	5	≥3,200	No data	0/4	0/4†	No isolate
3	*Macropus rufogriseus*	3 August 1999	5	No data	No lesions	4/4	0/4‡	Isolate lost
4	*Macropus rufogriseus*	23 July 2002	8	≥12,800	Brain	10/10	Not done	Tg WyUs 2
5	*Macropus rufogriseus*	23 July 2002	4	≥12,800	Brain	10/10	Not done	Tg WyUs 3

* Brain, lung, heart, and skeletal muscle.
† Liver, lung, and skeletal muscle.
‡ Liver.

lost her joey after 4 mo in the pouch; therefore, atovaquone was discontinued, resulting in a treatment duration of 6 mo. Wallaby no. 2 became too difficult to catch when her joey was 5 mo in the pouch. Atovaquone was discontinued at that time, having been given for 9 mo. That joey remained with the dam, rather than being hand raised and developed to independence without difficulty. Therefore, the duration of treatment with atovaquone was 9 mo for wallaby no. 2 and 6 mo for wallaby no. 3. Both females continued to be fertile and raised an additional joey.

Wallaby no. 2 died from sepsis due to development of lumpy jaw with secondary aspiration pneumonia approximately 2.5 yr after the toxoplasmosis was diagnosed. At necropsy, tissue samples were collected for bioassay in mice (Table I).

Wallaby no. 3 had to be killed due to development of acute uveitis accompanied by behavior consistent with pain and blindness 3 yr from the time toxoplasmosis was diagnosed. At necropsy, tissues were collected for bioassay in mice (Table I).

Wallaby no. 4, a male *M. rufogriseus*, was purchased from a breeder in Pennsylvania by C.C on 3 April 1995 when approximately 7 mo old. This wallaby was found to be shivering on 11 January 1997, at the age of 27 mo. Toxoplasmosis was suspected and was later confirmed by serology (Table II). Wallaby no. 4 was started on atovaquone 50 mg/kg, oral, twice daily, along with an equivalent volume of peanut oil. During the first week, the wallaby demonstrated weakness, lethargy, and loss of appetite. After 1 wk of treatment, his behavior seemed to be completely normal and the dose of atovaquone was reduced to 50 mg/kg, oral, daily. Treatment was continued for 2 mo. Wallaby no. 4 did not develop visual problems or abnormal ophthalmic signs. The wallaby was killed when 5 yr old, and tissues were collected for *T. gondii* isolation (Table I).

TABLE II. Serological responses of wallabies naturally infected with *T. gondii.*

Date bled	MAT titers of Bennett's wallaby no.			
	2	3	4	5
2 August 1996	<25	<25	<25	
18 September 1996	≥500	≥3,200	<25	
29 September 1996	≥3,200*	≥3,200	ND†	
27 October 1996	24,800	49,600	ND	
16 December 1996	24,800	24,800	ND	
13 January 1997	ND	ND	400	
31 January 1997	ND	ND	6,400	
16 February 1997	25,600	51,200	6,400	
5 April 1997	12,800	25,600	≥102,400	
4 May 1997	12,800	102,400	ND	
27 June 1997	6,400	102,400	ND	
27 July 1997	ND	102,400	ND	
7 December 1997	204,800	409,600	409,600	
30 April 1998	25,600	≥500	ND	
8 November 1998	25,600	≥500	≥500	
1 March 1999	≥3,200	ND	ND	
27 November 1999			ND	<25
23 April 2000			≥3,200	<25
23 July 2002			≥12,800	≥12,800

* ≥ = end titration not done.
† ND = no data.

Wallaby no. 5, male *M. rufogriseus*, was born in Pennsylvania (dam, wallaby no. 2 and sire, wallaby no. 4) on 20 August 1998. The joey was hand raised after 6 mo of age. At 9 mo of age, the joey collapsed and was unable to get to his feet. He was found to have spots of yellow exudate on ophthalmic examination, clinical signs consistent with toxoplasmosis. Wallaby no. 5 was treated with atovaquone 300 mg/kg, and canola oil (canola oil was used rather than peanut because the lipid composition is closer to that of marsupial milk, G. Smith, pers. comm., developer and manufacturer of Biolac For Marsupials, milk replacement formula, 5 May 1999) 4 ml orally, along with decadron 2 mg/kg, intramuscular injection, on the initial day. Treatment was continued for the next 6 wk with canola oil and atovaquone 100 mg/kg, oral. The dose was not increased appropriately for the wallaby's increasing weight during the subsequent 7 wk. Cloudiness of the vitreous was then found by ophthalmic examination and atovaquone 100 mg/kg, oral, was given twice daily for the remaining 3 mo of treatment. A single dose of decadron, 1 mg/kg, intramuscular injection, was given 1 wk after the atovaquone dose was increased. The clouding of the vitreous cleared subsequently. Inflammation of the vitreous 2 yr later was treated by atovaquone 50 mg/kg, oral, twice daily, along with canola oil, for 1 mo. Serology supported the clinical impression of toxoplasmosis (Table II). The wallaby was killed when 4 yr old, and tissues were collected for *T. gondii* isolation (Table I).

Serological examination

Wallabies were sedated with diazepam, 0.5 to 1 mg/kg (Spielman, 1992), and blood samples were collected from them at intervals (Table II). These serum samples were tested at APDL for antibodies to *T. gondii* using the modified agglutination test (MAT) as described previously (Dubey and Desmonts, 1987).

Bioassay for *T. gondii*

Wallabies were killed by an overdose of phenobarbitol. Attempts were made to isolate *T. gondii* from all 5 wallabies at APDL. Their tissues (brain separately and other tissues pooled) were homogenized in 10 volumes of 0.85% aqueous NaCl (saline); filtered through gauge; centrifuged; decanted; and a portion of the sediment was suspended in antibiotic saline (1,000 units of penicillin and 100 μg of streptomycin per milliliter of saline), and then an aliquot was inoculated subcutaneously into Swiss Webster (SW) mice from Taconic Farms (Germantown, New York) (Table I).

The mice inoculated with wallaby tissues were examined for *T. gondii* infection. Imprints of lungs and brain of mice that died were examined for *T. gondii* tachyzoites or tissue cysts. Survivors were bled on day 40–42 postinoculation (PI) and a 1:25 dilution of serum from each mouse was tested for *T. gondii* antibodies with the MAT. Mice were killed 45–48 days PI, and brains of all mice were examined for tissue cysts as described previously (Dubey and Beattie, 1988). The inoculated mice were considered infected with *T. gondii* when tachyzoites or tissue cysts were found in tissues.

To obtain oocysts of the *T. gondii* strain from wallaby no. 1 (Table I), infected mice were fed to a *T. gondii*-free cat. Feces of the cat were examined for shedding of *T. gondii* oocysts 3–14 days postingesting infected tissues as described previously (Dubey, 1995). Fecal floats were incubated in 2% sulfuric acid for 1 wk at room temperature to allow sporulation of oocysts, and they were bioassayed orally in mice (Dubey and Beattie, 1988). Sporulated oocysts were counted and diluted 10-fold 10^{-1} to 10^{-6}, and aliquots from each dilution were fed to 5 mice. The mice fed oocysts were examined for *T. gondii* infection.

Immunohistochemical staining

Specimens of cerebrum, cerebellum, pons, medulla, liver, heart, tongue, leg muscle, kidneys, spleen, small intestine, and lungs of 3 wallabies (nos. 3–5; Table I) were fixed in 10% formalin, and sections were processed routinely, sectioned, stained with hematoxylin and eosin, and examined microscopically. Deparaffinized sections were stained with polyclonal sera to *T. gondii* as described previously (Lindsay and Dubey, 1989).

Genetic characterization for *T. gondii*

The DNA was extracted from the tissues of infected mice (Lehmann et al., 2000). The restriction fragment length polymorphism (RFLP) strain type of *T. gondii* isolates was determined by nested polymerase chain reaction (PCR) on the SAG2 locus according to Howe et al. (1997).

RESULTS

Wallaby no. 1 had an MAT titer of 1:100, 14 days before death, and 1:200 on the day of necropsy (Table II). The remaining 4 wallabies were seronegative on their first sampling and became seropositive; they remained seropositive for the remainder of their lives (Table II).

Microscopic lesions were found in the brains of wallaby nos. 4 and 5 and were characterized by mild gliosis and perivascular infiltrations with mononuclear cells in cerebrum. *Toxoplasma gondii* was not found in histological sections stained with hematoxylin and eosin or with *T. gondii* antibodies.

Toxoplasma gondii was isolated from tissues of 4 of the 5 wallabies (Table I). Three of the 4 isolates were designated Tg WyUs 1–3; the fourth isolate was lost and not cryopreserved. Genotyping of these 3 *T. gondii* isolates using PCR-RFLP marker SAG2 revealed that 3 of the 4 isolates were genotype III.

The mice inoculated with wallaby tissues remained asymptomatic, indicating that tachyzoites and bradyzoites of these isolates were nonpathogenic for mice. However, all mice fed 10^{-1} to 10^{-3} dilutions of oocyst suspension from isolate from wallaby no. 1 died of acute toxoplasmosis 7–12 days later. The mice fed 10^{-4} dilution developed neurological signs and had to be killed 5 mo later. One of the 5 mice fed the 10^{-5} dilution of oocysts became infected, and numerous tissue cysts were found in its brain. The 10^{-6} dilution of oocysts was not infective to mice. Thus, <10 infective oocysts were pathogenic to SW mice.

DISCUSSION

The ingestion of oocysts from the environment is the most likely source of infection for wallabies. The problem this presents is that it usually is difficult, if even possible, to determine whether there was contamination of foods given or grazed, or whether oocysts were acquired from soil ingested during grooming. The exact time or time frame when oocysts were consumed, the number ingested, and any subsequent episodes or periods of time when oocysts were ingested are unknown. Furthermore, the time period required after infection and before observable signs of illness is unknown, along with the influence of variables such as the number of oocysts ingested, health, age of the wallaby, or environmental conditions. Accordingly, whether the wallabies nos. 1–3 in the present study acquired *T. gondii* infection in the United States or in New Zealand is not known.

Although wallabies nos. 4 and 5 acquired *T. gondii* infection in the United States, it cannot be determined how long they were infected before demonstrating signs of illness. Wallaby no. 4 had fewer signs than the rest. The effect of the young age of wallaby no. 5 relative to severity of initial signs and development of seroconversion cannot be determined. In that case alone, in retrospect, it was determined that the wallaby had some opportunity to eat hay harvested from a field where cat litter was later found to have been dumped. Hay for wallaby use was harvested from other fields and stored separately, in a loft inaccessible to cats, but contamination by feral cats could not be prevented before storage.

Infected mammals generally develop immunoglobulin (Ig)M before the development of IgG. The MAT detects only IgG antibodies because the mercaptoethanol used in the test destroys IgM and IgM-like substances that interfere in the test (Dubey and Desmonts, 1987). Therefore, a single high titer in this test does not mean recent infection because the time of onset of IgG development in wallabies is largely unknown. Under controlled conditions, 2 *M. giganteus* fed 50 or 500 *T. gondii* oocysts and 1 inoculated intramuscularly with 250 tissue cysts developed IgG antibodies by 3 wk PI using the MAT (Johnson et al., 1989). Four Tasmanian pademelons (*Thylogale billardierii*) with histologically confirmed acute toxoplasmosis had IgM, but not the IgG antibodies to *T. gondii* (Johnson et al., 1989). However, we stress that not all macropods are equally susceptible to toxoplasmosis. The smaller species, traditionally referred to as wallabies, demonstrate a vulnerability to toxoplasmosis that is not apparent in the larger species, commonly referred to as kangaroos.

In the present study, wallaby no. 1 had an MAT titer of 1:100 14 days before death, but the significance of this was not appreciated at the time. Wallabies nos. 2 and 3 had no detectible IgG antibodies in 1:25 serum dilution when first tested on 2 August 1996. Within 2 mo, both wallabies had high titers by the MAT, and these titers remained elevated for >12 mo, in spite of antiprotozoal therapy (Table II). Before this, it had not been possible to follow serology from wallabies infected with toxoplasmosis over such extensive time because they never survived.

Toxoplasmosis has been reported from many species of Australian marsupials, but little is known of the biological characters of *T. gondii* from these hosts. Johnson et al. (1988) isolated *T. gondii* by mouse inoculation from the brains of 6 of 17 *M. rufogriseus* and 4 of 17 Tasmanian pademelons (*T. billardierii*) from Tasmania, Australia. These animals were killed as part of a crop protection program, and their clinical status was not stated (Johnson et al., 1988). There is only scant information concerning isolation of *T. gondii* from wallabies from other countries. Mandelli et al. (1966) isolated viable *T. gondii* in mice inoculated from tissues of 3 Bennett's kangaroos (*Macropus bennetti*) that died in a zoo in Milan, Italy. Dobos-Kovács et al. (1974) fed tissues of 2 derby kangaroos (*T. eugenii*) to a cat and the cat subsequently shed *T. gondii* oocysts; the kangaroos had died in a zoo in Budapest, Hungary. Basso et al. (2007) isolated *T. gondii* from 2 captive *M. rufogriseus* in Argentina. The present report is the first isolation of *T. gondii* from *M. rufogriseus* and *M. eugenii* in the United States.

Why wallabies are highly susceptible to clinical toxoplasmosis is not known. Morphologically and biologically there is only 1species, *T. gondii* in the genus. Howe and Sibley (1995) genetically grouped *T. gondii* isolates from animals and humans into 3 genetic types (I, II, and III). Until that time, *T. gondii* isolates had been distinguished as mouse virulent or avirulent. Howe et al. (1997) suggested that type I isolates were highly pathogenic for mice, irrespective of the dose, and types II and III were relatively avirulent for mice. However, mouse virulence of *T. gondii* may have no correlates with clinical toxoplasmosis in higher animals and pathogenicity can vary with the host, the stage ingested, and the dose (Dubey, 2006).

It has been also proposed that all strains of *T. gondii* should be regarded pathogenic, irrespective of the mouse virulence (Dubey and Beattie, 1988). The dose, stage of the parasite ingested, and the immune status of the host influence the final clinical outcome of toxoplasmosis. Wallabies inoculated with a nonpathogenic, nonpersistent vaccine strain of *T. gondii* (S48) died of acute toxoplasmosis (Lynch et al., 1993). In the present study, tissue stages (tachyzoites and bradyzoites) of the *T. gondii* strains from wallabies caused asymptomatic infection in mice, whereas less than 10 oocysts of 1 isolate either killed the mice or made them ill. The isolates of *T. gondii* from all 3 wallabies that had clinical toxoplasmosis were genotype III. Nothing is known of the genetic diversity of *T. gondii* isolates circulating in various species of marsupials in Australia.

Treatment of wallabies with atovaquone seems to be effective therapy for toxoplasmosis. Atovaquone, a hydroxynaphthoquinone, is an antimicrobial (*Pneumocystis carinii* and an antiplasmodial) compound that has activity against both tachyzoites and tissue cysts in mice infected with *T. gondii*, and it has a synergistic effect in combination with pyrimethamine and clindamycin or sulfadiazine (Araujo et al., 1991, 1992, 1993; Djurkovic-Djakovic et al., 1999; Moshkani and Dalimi, 2000). It has been used to treat toxoplasmosis in acquired immunodeficiency syndrome (AIDS) patients who were allergic to sulfonamides or pyrimethamine and those with *T. gondii*-associated retinochoroiditis (Kovacs et al., 1992; Katlama et al., 1996; Pearson et al., 1999; Remington et al., 2001). In AIDS patients, 50% of persons treated with atovaquone relapsed with toxoplasmosis. Results of the present study indicate that the drug was well tolerated in wallabies. Histological and bioassay results indicated that in at least 1 wallaby treatment with atovaquone killed *T. gondii* and reduced damage to the host because lesions were not seen. In another wallaby (no. 4), treated for only 2 mo, only mild lesions were detectable. The present study provided a rare opportunity to document efficacy of anti-*T. gondii* therapy in a wild animal species by postmortem examination involving immunohistology and bioassay. It is worth noting that the dose that was received by the wallabies is approximately 10 times the amount customarily used to treat humans, when compared on the basis of milligrams per kilogram.

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ERRATUM . . .

We would like to correct an error in our article (J. P. Dubey, J. G. Humphreys, and D. Fritz, 2008, ‘‘A new species of *Sarcocystis* (Apicomplexa; Sarcocystidae) from the black bear (*Ursus americanus*)’’; Journal of Parasitology 94: 496–499):

Under ‘‘*Specimens deposited*’’ the text reads, ‘‘Two histological sections stained with H and E were deposited as syntypes for bears nos. 1 and 2 in the United States National Parasite Collection (USNPC nos. 10101 and 10102), United States Department of Agriculture, Beltsville, Maryland. One H and E stained section from bear no. 3 was deposited as voucher specimen from bear no. 3 (USNPC no. 10103).’’ The USNPC numbers should have been 100101–100103.